Interface of epilepsy and sleep disorders

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Obstructive sleep apnoea was first brought to prominence by Henri Gastaut, a French epileptologist. Since that time the interface between epilepsy and sleep disorders has received less attention than might be justified, recognizing that sleep deprivation is a poignant provocateur for seizures. Sleep deprivation is often used as a diagnostic procedure during electroencephalography (EEG) when waking EEG has failed to demonstrate abnormality. Patients referred to an outpatient neurological clinic for evaluation of possible seizures in whom sleep disorder was suspected, either due to snoring during the EEG or based on history, were evaluated with all-night diagnostic polysomnography (PSG) and appropriate intervention administered as indicated. Patient and seizure demography, sleep disorder and response to therapy were reviewed and the interface explored. Fifty patients aged between 10 and 83 years underwent PSG. Approximately half were diagnosed with epilepsy and almost three-quarters had sleep disorders sufficiently intrusive to require therapy (either continuous positive air pressure (CPAP) or medication). With co-existence of epilepsy and sleep disorders, proper management of sleep disorders provided significant benefit for seizure control. Snoring during EEG recordings could alert to the possibility of a sleep disorder even with epilepsy diagnosed. Where both epilepsy and sleep disorder coexist appropriate management of the sleep disorder improves control of the epilepsy.

Key words: sleep disorders; epilepsy; apnoea; diagnosis; polysomnographs.

INTRODUCTION

The most commonly acknowledged interface between epilepsy and sleep is the recognition that seizures may be more common during sleep and within the period immediately following waking[1–4]. Sleep deprivation has been used for many years as a powerful provocateur for diagnostic electroencephalography (EEG) in suspected cases of epilepsy in which the standard waking EEG study has failed to demonstrate abnormality[5–7].

There is an appreciation that epilepsy may be exacerbated by sleep deprivation[4–11]. Recently, there have been reports which indicate that various types of epilepsy are being misdiagnosed as sleep disorders[12–15] reinforcing the need for neurologists to have a greater understanding of both epilepsy and sleep disorders, so that such misdiagnoses can be avoided in the future. It is also recognized that sleep disorders may resemble seizures[16] further emphasizing the need to consider both diagnoses in patients referred for either diagnosis.

Epilepsy is reported to occur in as many as 2% of the population when allowance is made for the many confounding variables[17]. The widely quoted prevalence rate for epilepsy is between 0.5–1%[18]. Amongst the most common sleep disorders to be encountered, are sleep apnoea (SA), periodic limb movement in sleep (PLMS) and upper airway resistance syndrome (UARS)[19]. The epidemiology of sleep disorders, and particularly SA, is far from straightforward[20–26] with patients complaining both of insomnia and hypersomnalence as presenting features thereof [26].

The frequency of SA varies between studies with prevalence figures of 18% to 73% amongst the elderly[24] being reported as having SA. Two respected surveys quoted prevalence figures for SA of approximately 40% for healthy elderly people[27, 28]. Fleury[24] considered 18% to be a more acceptable figure. Partinen and Telakivi[22] stated that the highest prevalence figure for SA in the elderly was approximately 25% for the over 65-year-olds who had a respiratory disturbance index of 5 or more. They also reported a minimum prevalence of SA in adult men of approximately 1%, a figure supported by Phillips *et al.*[23] and Soldators and Lugaresi[25]. Hypersomnalence is the more common presentation of SA[19] but is also reported in patients with epilepsy[29, 30]. PLMS is also a common sleep disorder which has been reported to occur in approximately 6% of a control group of healthy medical centre personnel[26, 28] with figures as high as 45% for people aged

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Table 1: Patient demographics.

Age at sleep study	Gender	Body mass index	Referral reason	Epilepsy	Sleep study result	Intervention required	CPAP continued
27	Female	–	? epilepsy	No	Hypopnea PLMS	CPAP	Yes
27	Male	29.3	2nd opinion	Yes	Negative	–	–
27	Male	36.3	2nd opinion	Yes	UARS Sleep apnoea	CPAP	Yes
51	Male	28.7	2nd opinion	Yes	Sleep apnoea	CPAP	Yes
10	Male	–	? epilepsy	Yes	PLMS (v. mild)	No	–
27	Male	29.7	2nd opinion	Yes	UARS (v. mild)	No	–
66	Male	33.2	2nd opinion	Yes	Negative	–	–
72	Female	30	? epilepsy	Yes	Sleep apnoea PLMS	CPAP	Yes
34	Male	–	? epilepsy	Yes	Hypopnea PLMS	CBZ	–
51	Male	35.9	? epilepsy	No	Hypopnea	CPAP	No
27	Male	26.4	? epilepsy	No	Negative	–	–
47	Male	31.6	? epilepsy	No	Sleep apnoea UARS/PLMS	CPAP	Yes
58	Male	26	? epilepsy	No	Sleep apnoea (v. mild)	No	–
64	Male	28.4	? epilepsy	No	Hypopnea Sleep apnoea	CPAP	No
65	Female	37.3	? epilepsy	No	Hypopnea Sleep apnoea	CPAP	Yes
63	Male	–	? epilepsy	No	Hypopnea (v. mild) Sleep apnoea (v. mild)	No phenytoin	–
52	Male	–	? epilepsy	No	PLMS UARS	Referral respiratory physician	–
53	Male	36.1	? epilepsy	No	Sleep apnoea	CPAP	Yes
62	Female	30.5	? epilepsy	No	Sleep apnoea (v. mild)	No Weight reduction	–
42	Male	24.7	2nd opinion	Yes	Negative	–	–
51	Male	30.9	? epilepsy	No	Sleep apnoea Hypopnea	No Weight reduction then ? CPAP	–
70	Female	–	2nd opinion	Yes	Hypopnea	CPAP	No
47	Male	26.7	2nd opinion	Yes	Hypopnea (v. mild)	No	–
57	Female	30.4	? epilepsy	No	Sleep apnoea UARS	CPAP	No
45	Female	30.1	? epilepsy	Yes	UARS PLMS	Referral respiratory physician and Sinemet	–
42	Female	19.4	? epilepsy	No	Negative	–	–
38	Male	–	2nd opinion	Yes	Sleep apnoea	CPAP	Yes
46	Male	22.5	2nd opinion	Yes	Negative	–	–
38	Female	40.6	? epilepsy	No	Negative	–	–
45	Male	30.8	? epilepsy	No	UARS Sleep apnoea	CPAP	No
48	Male	–	? epilepsy	No	Hypopnea Sleep apnoea	CPAP	No
27	Male	–	2nd opinion	Yes	Negative	–	–
43	Female	–	? epilepsy	No	PLMS	Sinemet	–
49	Male	29	2nd opinion	No	UARS/PLMS	Sinemet	–
43	Male	23.8	? epilepsy	Yes	Sleep apnoea	CPAP	Yes
44	Male	25.1	2nd opinion	Yes	UARS PLMS	Sinemet	–
21	Female	47.9	2nd opinion	Yes	Hypopnea	CPAP Weight reduction	No
58	Male	31.1	? epilepsy	Yes	UARS/PLMS Sleep apnoea	Sinemet CPAP	No

Table 1: Continued.

Age at sleep study	Gender	Body mass index	Referral reason	Epilepsy	Sleep study result	Intervention required	CPAP continued
44	Male	30.6	? epilepsy	No	UARS	Referral respiratory physician	–
76	Male	25.9	? epilepsy	No	Sleep apnoea	CPAP	No
41	Female	48.2	? epilepsy	No	Sleep apnoea Hypnopnea	CPAP Weight reduction	No
20	Female	–	? epilepsy	No	Sleep apnoea PLMS	CPAP	Yes/No
32	Male	–	? epilepsy	Yes	Sleep apnoea	CPAP	Yes
83	Male	–	2nd opinion	Yes	Sleep apnoea PLMS	CPAP Sinemet	No
39	Female	32.5	2nd opinion	Yes	Sleep apnoea Hypopnea/PLMS	CPAP	No
54	Male	24.4	2nd opinion	Yes	Sleep apnoea	CPAP	Yes
67	Male	28.3	? epilepsy	No	Sleep apnoea UARS, PLMS	CPAP	No
51	Male	32.0	? epilepsy	No	Sleep apnoea UARS, PLMS	CPAP	No
20	Male	26.0	2nd opinion	Yes	Worth Considering CPAP	CPAP	No
53	Female	16.3	? epilepsy	No	UARS	CPAP	No

PLMS, periodic limb movement in sleep; UARS, upper respiratory airway syndrome; CBZ, carbamazepine.

65 years and older having a myoclonus index of 5 or more[27, 31].

In recent times there has been a growing interest in the relationship between sleep disorders and epilepsy with an appreciation that the correct management of the sleep disorders may positively benefit the management of the epilepsy[32–35]. These publications each focused on very small numbers of patients who have epilepsy (each between four and 10 patients), thereby demonstrating a potential bias in sample selection and possible problems for extrapolation to a wider epileptic community.

The paper that follows reports the findings in a sample of 50 people referred to a private sleep laboratory from a centre which specializes in the management of those with epilepsy.

MATERIALS AND METHODS

Patients who were referred to a sleep laboratory from a neurological out-patient clinic which specializes in the management of people with epilepsy between September 1991 and March 1997 had their records reviewed. Only those patients who were referred to the clinic for the evaluation of epilepsy at the time of initial presentation, were included within the study. These patients were assessed clinically by means of history, examination and suitable investigation, to determine the validity of the diagnosis of epilepsy, which was the primary cause for referral.

Patients who underwent EEG and who demonstrated early-onset snoring, during either sleep-deprived EEG or a standard waking study, were referred to the St Lukes Sleep Centre for all night-diagnostic polysomnographic (PSG) study. Other patients referred for assessment of epilepsy but in whom the history was suggestive of SA or in whom other diagnostic procedures, such as video telemetry, indicated the potential for SA were also referred for similar PSG investigation.

Parameters measured in PSG studies included: EEG (using C_3-A_2 O_2-A_1 or transverse run from vertex using 10–20 international placement of electrodes); EOG (with electrodes at the outer canthus muscle, right upper and left lower); EMG (from chin and tibialis anterior); nasal/oral airflow (employing naso–oral thermocouple); respiratory effort (adopting piezoelectric technology with abdominal and thoracic belts); oximetry; and ECG (by means of modified rhythm strip). The PSG were recorded using Biologic Sleepscan computerized equipment with each PSG study being manually scored by a qualified sleep technician.

The data from these studies were reviewed by an independent researcher experienced in projects dealing with both epilepsy and sleep disorders. The diagnoses were determined and compared with the presenting diagnosis at the time of referral to the epilepsy clinic. These data were then compiled and analysed to assess the interface between the two diagnoses.

RESULTS

Patient demographics

One hundred and twenty-eight files of patients referred to the sleep laboratory between September 1991 and March 1997 by the neurology clinic, were reviewed to ensure that only those who were thought to have epilepsy at the time of initial assessment at the clinic were included in the study. This excluded those pa-

tients referred for headache, excess fatigue or for specific sleep analysis. This produced a sample of 50 patients initially thought to have epilepsy by the referring doctor. The age range of the 50 patients was 10–83 years (mean 46 ± 16), comprising 35 males and 15 females (Table 1). Ten of the patients (20%) were older than 60 years. Body mass index (BMI) was available for 37 (74%) patients of whom 19 (51%) had a BMI $\geq$ 30 kg m^{-2} and only two patients had a BMI $<$ 20 kg m^{-2}. The range of available BMI was 16.3 to 48.2 kg m^{-2} (mean 30.2 ± 6.5) (Table 1). Reason for referral for PSG was snoring during the EEG for 32/50 (64%) with the remainder being selected consequent to a high index of suspicion.

Sleep studies

Forty-two of 50 patients (84%) had some form of sleep disorder with only 8/50 (16%) demonstrating a completely negative PSG. Thirty-six of 50 patients (72%) showed sufficient abnormality to require intervention (although this included a weight reduction program for three patients). With BMI $\geq$ 30 kg m^{-2}, 17/19 (89%) had sleep disturbance sufficient to require intervention while 10/18 (56%) of those with BMI $<$ 30 kg m^{-2} required intervention for sleep disorder (Table 1). Of the 32 who were referred because of snoring during the EEG, 26/32 (81%) had positive PSG, comprising 17 with SA, seven with hypopnea, 10 with UARS and eight with PLMS. Twenty-nine of 35 (83%) males and 13/15 (87%) females had positive PSG (Table 1). With regard to age, 9/10 (90%) of the over 60-years olds had sleep disorders of whom eight required intervention. Thirty-three of 40 (83%) of the under 60-years-olds had positive PSG of whom 27 (68%) required intervention which was more significant than recommending weight reduction.

Sleep disorders

Twenty-seven of 50 patients (54%) had SA, 13/50 (26%) had hypopnea and 14/50 (28%) had UARS indicating that a significant number of patients had respiratory causes of disturbed sleep (Table 1). Twenty-six of 50 patients (52%) had pure respiratory disturbance without PLMS also contributing some degree of sleep disturbance as well. Of the 42 patients with some form of sleep disorder only 17 (40%) had a single pure diagnosis with the majority having more than one type of disturbance. Sixteen of 50 patients (32%) had PLMS of whom six had sufficient PLMS to justify medication, predominantly combination of levodopa with carbidopa (Sinemet), to be prescribed.

Of the 24/50 patients (48%) who were confirmed to have epilepsy, 19 (79%) also had sleep disorders, thereby confirming that 23 (55%) of those diagnosed as having sleep disorders were found not to have epilepsy, despite it being the cause for referral to the neurology clinic. Eleven of 28 patients (39%) prescribed CPAP, were actually using it at the time of the study. Some said that they would use it; have a CPAP 'holiday' and return to its use. Fifteen of 28 (54%) prescribed CPAP did not continue its use, either due to emotional factors (3), mask intolerance (4), lack of efficacy (2), or lost to follow-up (6).

Outcome measures

Of those in whom intervention was prescribed outcome measures were determined for 32/36 (89%) of whom 16 (50%) reported improvement (Table 2). It was difficult to obtain absolute seizure numbers but where available six (19%) reported being seizure-free or having a >50% reduction in seizure rate (Table 2).

DISCUSSION

Of those patients referred to a sleep laboratory by a neurological clinic known to have an interest in epilepsy ($n = 128$), 50 patients were initially referred to the neurological clinic with a suspected diagnosis of epilepsy. Of these 50 patients, 18 were considered definitely to have epilepsy at the time of referral with 32 probable epilepsy. After a full evaluation, only 24/50 (48%) retained the diagnosis of epilepsy and of them 19 (79%) also had sleep disorders, thereby reinforcing the need for those who manage patients with epilepsy to have a high index of suspicion for sleep disorders. This is especially so with the elderly patients, those with a BMI $\geq$ 30 kg m^{-2} and those who snore during the EEG recording.

This sample showed a specificity of 84% (42/50) with positive yield from referral to a sleep laboratory, based upon the selection criteria of snoring during EEG (32/50, 64%) and upon the seeking of appropriate history while evaluating patients who were initially thought to have epilepsy.

These findings support earlier studies which report increased sleep disorders among the elderly and the obese, irrespective of the concomitant diagnosis of epilepsy[20–25]. The reported increase of SA among males was not found in this sample population although this may merely reflect the relatively small number of females in the sample. Of greater importance is the fact that within this sample, only approximately half the patients retained the diagnosis of epilepsy after full evaluation.

CPAP was prescribed for 56% of the population with

Table 2: Treatment outcome in patients requiring CPAP/Sinemet.

CPAP/Sinemet required	CPAP continued	Outcome	Epilepsy diagnosis
CPAP	No	Psychological antipathy to CPAP	No
CPAP	Yes	Sleeping better, more refreshed, decrease in 'funny turns'	No
CPAP	Yes	Improvement	Yes
CPAP	Yes	>50% reduction in seizure frequency, feeling better	Yes
CPAP	Yes	Sleeping better, no improvement in seizure control	Yes
CPAP	No	Stopped CPAP as no improvement, though snored less	No
CPAP	No	Mask uncomfortable, patient did not see benefit	Yes
Sinemet	–	Seizure free, improvement with Sinemet	Yes
CPAP	Yes	>50% reduction in seizure frequency	Yes
CPAP	Not applicable	Still waiting for CPAP study, treatment not initiated	No
CPAP	Yes	More alert and talkative, less frequent/severe seizures	Yes
CPAP	Yes	Using CPAP with good effect, patient more alert	No
Sinemet	–	Significant improvement	No
CPAP	Yes	>50% reduction in seizure frequency	No
CPAP	No	Lost to follow-up	No
CPAP	No	Mask intolerance, treatment not initiated	No
CPAP	Yes	Feels better, has more energy, seizure frequency unchanged	No
CPAP	Yes	Not on regular basis, >50% reduction in seizure frequency	Yes
Sinemet	–	Significant improvement	No
Sinemet and CPAP	Not applicable	Sleeping better and longer on Sinemet, CPAP treatment not initiated	Yes
Sinemet	–	Feeling terrific on Sinemet	Yes
CPAP	No	Uncomfortable, unable to tolerate mask	No
CPAP	Yes/No	Machine broke after 1 year, carers saw no benefit	No
CPAP	No	Lost to follow-up	No
CPAP	Yes	Seizure free, more alert and refreshed	Yes
Sinemet and CPAP	No	Patient unable to tolerate mask, improvement on Sinemet	Yes
CPAP	No	Lost to follow-up	Yes
CPAP	No	CPAP on hold (trying weight reduction first)	Yes
CPAP	No	Lost to follow-up	No
CPAP	No	Had CPAP titration, treatment not initiated	Yes
CPAP	No	Lost to follow-up	No
CPAP	No	Lost to follow-up	No

sleep disorders but less than half of them continued to use the equipment, thereby demonstrating a significant attrition/compliance problem with this form of intervention.

Three patients, all with UARS, required involvement of a respiratory physician, thereby demonstrating that neurologists can manage the majority of those patients who have sleep disorders.

It has already been established that sleep deprivation can negatively impact upon epilepsy[4–11], hence it follows that intervention to improve quality of sleep should have a positive effect upon seizure control. This hypothesis was confirmed in this study of patients with epilepsy, where 6/32 (19%) had $\geq$50% reduction of seizures and of whom two were seizure-free. A total of 16 patients reported positive gains from intervention for their sleep disorder despite the fact that follow-up was limited and compliance with therapy, such as use of CPAP, was found to be suboptimal.

These data confirm the benefits of appropriate diagnosis and management of sleep disorders in both epileptic and nonepileptic patients and the need to evaluate patients referred with suspected epilepsy critically to ensure correct diagnosis and optimal therapeutic intervention.

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